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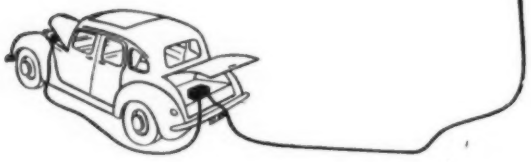

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Refresher Article No. 14

Foot-and-Mouth Disease

BY
A. M. GRAHAM
Ministry of Agriculture Animal Health Division, Woodcote, Oxon

OVER the years no disease of animals in this country has received greater publicity or aroused more interest, not to say controversy, than foot-and-mouth disease. The rapid and spectacular manner in which it spreads, the drastic and far-reaching methods taken to combat it, and the widespread dissemination of information about outbreaks in the press, on the radio and, latterly, on television, have all combined to keep it very much before the public. Yet despite all that has been said and written there still seems to be a lack of knowledge of the disease, and of the reasons for the slaughter policy which has been the method of dealing with outbreaks in this country for nearly 70 years. The purpose of this article is to give some general information about the disease, the symptoms and lesions it produces in farm animals, the procedure for dealing with outbreaks, the reasons for this procedure, and its importance not only to the livestock industry but to the veterinary profession upon whose vigilance and judgment the success of the slaughter policy so often depends.

History

The disease was first recorded in England in 1839 at Stratford. At that time the importation of live susceptible animals was prohibited, as it is to-day from most countries except in special circumstances, but the disease was prevalent on the Continent and was probably brought here by indirect means. Once it had gained a foothold it spread rapidly through many counties in England, eventually reaching Scotland and Ireland. Thereafter it continued to appear intermittently in this country, the most serious outbreak occurring in 1871, when about three million animals were said to be affected. It was this epidemic which resulted in the disease being made notifiable. The slaughter policy was introduced in 1892, and there were no outbreaks in the years 1895 to 1899, 1903 to 1907 and in 1917. With these exceptions outbreaks continued to occur annually, the peak years being 1922 (over 1,000 outbreaks), 1923 (about 2,000 outbreaks), 1924 (nearly 1,500 outbreaks) and in 1952 (approximately 500 outbreaks).

The Disease

Foot-and-mouth disease is one of the most infectious diseases known and can spread with amazing rapidity both by direct and indirect means. The animals susceptible are cattle, sheep, goats, swine, wild ruminants and, to a lesser extent, hedgehogs and rats. A few cases have been reported in humans, mainly abroad, but the evidence of human infection is not very convincing.

The disease is due to a virus which multiplies rapidly, particularly in the mucosa of the tongue, dental pad, lips and in the skin around the coronet, in the interdigital space and at the bulbs of the heels causing vesicles to develop on all or any of these sites. Occasionally vesicles are found on the teats and udder. These are the outward manifestations of the disease but the virus is also present in the tissues, organs, and body discharges.

There are several types of virus, O, A and C being those most commonly found in this country. Variant strains within these types also appear from time to time. An animal which has recovered from an attack with one type develops immunity to that type but is not protected against the others.

The incubation period is normally between 3 and 8 days but it can be shorter or longer and periods up to 2 to 3 weeks have been recorded. It is believed that the long incubation periods result from a small amount of infecting virus or, possibly, from a weak or attenuated strain.

The virus can multiply only in the living animal

but it can survive in dead tissue or in a favourable environment for long periods. It is quickly destroyed by heat, sunlight and disinfectants, but drying, cold and darkness favour its survival. Experimental work has shown that under suitable conditions it can remain viable for a least 14 days on wool, 4 weeks on cows' hair, 11 weeks on boot leather, 14 weeks on rubber boots and 15 weeks on hay. Cold storage conditions in the meat trade are also favourable to its survival, particularly in liver, kidney, tripe, bone marrow and lymph glands, but in muscle the production of acid associated with the onset of *rigor mortis* quickly inactivates the virus.

The disease is not usually fatal in adult stock but it can cause heavy mortality in young animals. The loss of condition, reduced milk yield, and the secondary consequences such as abortion, chronic lameness, sterility and mastitis, which may follow an attack of the disease, can cause crippling losses, particularly in dairy cattle. It is the cumulative effects of the disease and its great spreading propensities which make it such a menace to our highly susceptible livestock.

There are two main avenues through which infection reaches this country. South American meat is imported in large quantities to maintain our supplies and, although stringent precautions are taken in South America under the supervision of British veterinary officers to ensure that this meat is free from infection, it is not always possible to prevent the carcases of animals incubating the disease, yet showing no visible evidence of it, from being included in a consignment. Without doubt this meat is a source of outbreaks in this country, particularly in pigs which have access to unboiled swill.

Migrating birds are another means by which infection may reach these shores. The evidence against the birds, although largely circumstantial, is very strong. It has been shown experimentally, for example, that starlings can carry infection mechanically on their feet and feathers, and in their droppings following the ingestion of infective material. Starlings, seagulls and rooks have been blamed as well as other species, and outbreaks in this country, particularly in the eastern and south-eastern counties, often occur when bird migration coincides with a heavy incidence of disease on the Continent.

Airborne infection from the Continent is also a possibility and there may be other routes by which the disease is occasionally brought in, but there is overwhelming circumstantial evidence that contaminated South America meat and migrating birds are mainly responsible.

Symptoms

The classical symptoms of foot-and-mouth disease are salivation and lameness caused by the formation of vesicles in the mouth and on the feet. Generally these symptoms develop simultaneously, although on occasions, particularly in the early stages of the disease, only one site may be involved and there may be salivation with lameness and *vice versa*. The severity of the symptoms and the extent of the vesicle formation vary according to the character of the virus. With some viruses mouth lesions may predominate, and with others, foot lesions, but in the majority of cases the lesions are fairly evenly distributed on both sites. Before the vesication stage is reached there are usually some obvious signs of illness such as dullness, inappetence, perhaps uneasiness, a drop in milk yield in dairy cattle, pyrexia and sometimes shivering. This phase is short-lived, not more than a few hours, and as the vesicles develop is followed by quivering and smacking of the lips, salivation, difficulty in chewing, sometimes a slight nasal discharge, paddling, snatching, shaking and kicking out of the affected feet, marked lameness and loss of condition. The animal is in obvious pain, dejected and restless, but once the vesicles have ruptured the temperature usually drops and it appears to gain some relief and may move more freely. Vesicles may develop on the udder and teats of cows, and on rare occasions these may be the primary lesions giving rise to tenderness and soreness of the teats. In-calf cows may abort and young animals may die without showing any apparent lesions of the disease.

In sheep and pigs there is a preliminary stage of constitutional disturbance, with a high temperature, the animals lying about looking sick and in pain, followed by lameness, often on all four feet and disinclination to move. The affected animals, when made to rise, adopt a crouching attitude with the feet tucked under the body. There may be some salivation, but frequently this is not very noticeable in sheep and pigs, and the obvious symptoms are the sudden onset of acute lameness, the apparent sickness of the animal, its disinclination to feed and a falling off in condition. There are, of course, considerable variations in the extent and severity of the symptoms but the foregoing describes those usually found in typical cases of the disease.

Lesions and Diagnosis

The lesions of foot-and-mouth disease are very similar in character and distribution in cattle, sheep and pigs, although there are certain minor differences peculiar to each which will be referred to later.

In all three species vesicles form in the mouth and on the feet, and the first visible evidence of them is a blanching and under-running of the epithelium. These "blanched under-run" areas are the sites of rising vesicles which, as they develop and fill with lymph, cause raising of the overlying epithelium and form well defined and often prominent vesicular lesions. The vesicles closely resemble ordinary blisters and contain clear-coloured fluid. They vary in size from a pea to a golf-ball, or even larger if they coalesce, and when squeezed or pressed will rupture with escape of the contents, and collapse leaving the overlying epithelium loose and wrinkled. The epithelium is friable, soft and juicy and is easily picked off, all the layers down to the corium being involved. Usually the vesicles are fully developed within a few hours and, if they are not interfered with, will rupture of their own accord, probably as a result of internal or external pressure. The covering epithelium is quickly shed, except perhaps for small pieces which may adhere to the edge of the lesion for a time, but these are soon cast off and there are left behind denuded raw, red areas which

A. M. GRAHAM—FOOT-AND-MOUTH DISEASE

FIG. 1. Steer. Tongue. Developing vesicles raised and blanched.

FIG. 2. Steer. Dental pad. Developing vesicles raised and blanched.

FIG. 3. Steer. Tongue. Ruptured vesicles.

FIG. 4. Steer. Tongue. Recently ruptured vesicle.

FIG. 5. Steer. Interdigital space. Ruptured and unruptured vesicles.

FIG. 6. Steer. Bulb of heel. Ruptured and unruptured vesicles.

FIG. 7.—Steer. Tongue. Ruptured and unruptured vesicles.

FIG. 8.—Sheep. Dental pad. Recently ruptured lesion.

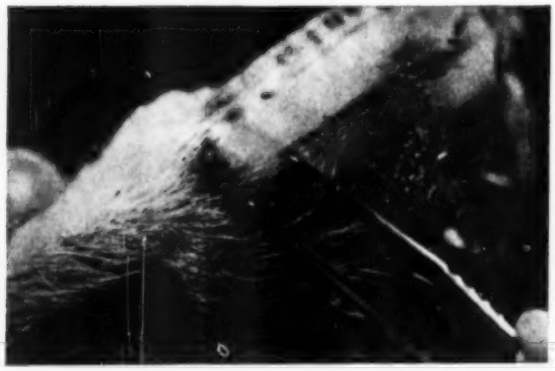

FIG. 9.—Sheep. Coronet. Unruptured vesicles.

FIG. 10.—Sheep. Interdigital space. Ruptured and unruptured vesicles.

FIG. 11.—Pig. Snout. Unruptured vesicle.

FIG. 12.—Pig. Coronet. Unruptured vesicles.

soon begin to heal. Provided there are no complications such as secondary infection, mouth lesions in cattle, for example, can heal completely in 10 to 14 days and the only visible evidence of their having existed is the lighter colour of the newly formed epithelium covering them.

The vesicles on the feet form in the skin of the interdigital space, at the bulbs of the heels, and around the coronet, and rupture fairly quickly as a result of movement and pressure. If there has been extensive vesication of the feet, particularly at the junction between the horn and the skin, separation of the horn from the skin and underlying tissue takes place and may extend right round the coronet. When this occurs, new horn is formed and gradually grows down the claw, replacing the old horn which is eventually shed. This process is often referred to as "thimbling." The extent of the growth of new horn is a valuable guide to the age of the lesions, particularly in those cases where there is reason to believe there may have been delay in reporting disease. Lesions do not necessarily develop on all the feet and, not infrequently, only one or two may be affected.

Cattle

In cattle the commonest sites for mouth lesions are the tip and dorsal surface of the tongue, the dental pad and inside the lips, although they can occur on the muzzle, hard palate and in the nostrils. They may be in different stages of development and their appearance varies with their age. There may be rising vesicles, fully developed unruptured vesicles, ruptured vesicles or areas completely denuded of epithelium in different stages of healing. All these variations may be seen in a single animal and, in the event of healing lesions only being encountered, it may be necessary to examine further animals in order to find more typical lesions. This should not be difficult for foot-and-mouth disease is highly infectious and soon reveals itself in other animals. In milking cows ruptured or unruptured vesicles may be observed on the udder and teats and, if so, a careful examination should be made of the mouth and feet of such animals. Many primary lesions of foot-and-mouth disease occur on the dorsum of the tongue and further back and, if the tongue is not fully withdrawn for examination, they may not be detected. The lesions are usually very painful and the animal resents handling.

The feet lesions in cattle are, as a rule, situated in the interdigital space and heel regions and, unless the feet are lifted or the animal cast and the feet washed, the lesions may easily be missed. In young stock, particularly calves, the lesions can be very indefinite and all that may be present may be congestion and abrasion of the buccal mucous membrane, and enteritis followed by death. If calves are sick or dying from some unidentified condition it is a wise precaution to inspect the nurse cows or other susceptible stock that have been in contact with them.

Sheep

Foot-and-mouth disease lesions in sheep are often not very obvious or extensive and a very careful examination may be necessary to detect them. The favourite site for the mouth lesions is the dental pad and, less frequently, the tongue and lips. Unruptured lesions on the dental pad are flat in appearance and may not be very easily seen. There appears to be under-running of the epithelium rather than vesication, and this type of lesion can best be demonstrated by pressing the finger or thumb over the surface of the dental pad and detaching the under-run epithelium. A similar type of lesion may be present on the tongue and lips.

The interdigital space, heel region and the coronet are the sites of the feet lesions, and they may not be very extensive, although they are usually fuller and more vesicular in character than the mouth lesions. Foot-rot may be co-existent with foot-and-mouth disease and can be a complicating factor, particularly as treatment given for the former tends to mask the foot-and-mouth lesions. In such cases a number of sheep may have to be examined before definite evidence of the disease is obtained.

Pigs

Foot-and-mouth lesions in pigs may be present on the snout and the tongue, but the feet lesions are those on which diagnosis is usually based. These lesions occur in the interdigital space, at the bulbs of the heels, around the coronet and sometimes on the supernumerary digits. If vesication of the feet is extensive complete separation of the horn at the top of the hooves from the skin and underlying tissues may take place. This is followed in a few days by the formation of new horn which gradually grows down from the coronet, replacing the old horn which is eventually shed—a process referred to earlier as thimbling and occasionally seen in pigs and sometimes in sheep. In pigs, as in sheep, sickness, sudden lameness, reluctance to rise and evidence of pain when moved are characteristic symptoms of foot-and-mouth disease, and these, combined with the lesions described above, should enable a diagnosis to be made or at least arouse suspicion and cause action to be taken.

It is not proposed to go into the question of differential diagnosis in this article for it is not practicable or desirable to attempt to give a detailed description of the various conditions simulating foot-and-mouth disease which can exist in cattle, sheep and pigs. The point that must be stressed is that, if animals show symptoms or lesions affecting the mouth or feet or both, the possibility of their being affected with foot-and-mouth disease should at once come to mind and a thorough examination should be made. Cases can be missed, particularly in the early stages, if this is not done, and missed cases can cause great harm.

The Disease in Practice

The practising veterinary surgeon is often the first person called in to see these cases and he is our first line of defence against the disease. If he has the slightest doubt about the animals, and is not completely satisfied that the condition is not foot-and-mouth disease, he should immediately get in touch with his Animal Health Divisional Office and ask

for a second opinion, remaining on the farm and ensuring that precautions are taken to prevent the spread of infection until a Veterinary Officer arrives. The latter will examine and discuss the case with him and, if it is negative, no further action need be taken; but should it prove to be positive, the machinery for dealing with outbreaks is immediately put in motion. This consultation procedure obviates the need for full-scale emergency measures in those cases where, in the practitioner's view, there are insufficient grounds for reporting suspected disease but an element of doubt still remains in his mind as to the nature of the condition. Of course, if the veterinary surgeon suspects that foot-and-mouth disease exists, he should act immediately, informing the Divisional Office and the police of his suspicion by telephone, and instructing the owner that there must be no movements of persons, animals or things on to or off the premises. The latter should literally be closed up until the Veterinary Officer arrives and every possible precaution taken to contain the infection. The important thing to remember is that, in either case, it is essential to act at once for foot-and-mouth is one of the most infectious diseases known and prompt action may not only prevent local spread but the widespread dissemination of infection through the movement of animals, persons or other agencies.

Suitable protective rubber clothing and rubber boots should always be worn when dealing with suspected cases of the disease or visiting farms in foot-and-mouth disease infected areas, and great care should be taken to ensure that the hands, nails, protective clothing, boots and any instruments or equipment used on the premises, are thoroughly disinfected on the completion of the examination or visit. Infection can readily be carried on the person, car and equipment, and experience has shown that the dangers of transmission by these means are very real indeed, particularly on the same day following a visit to a positive case.

Procedure for Dealing with Outbreaks

Whenever foot-and-mouth disease is suspected to exist on any premises they are immediately put under restrictions and the Veterinary Officer makes a diagnostic visit. If disease is confirmed the affected stock, and those in contact with them or otherwise exposed to infection, are valued and slaughtered and the carcases cremated or buried. A preliminary disinfection is carried out and is followed by a thorough disinfection of the whole premises as soon as the slaughter has been completed. The Infected Place restrictions prevent the movement off or on to the premises of persons, animals or things, unless specially authorised, and then only after adequate disinfection.

An Infected Area, covering a radius of about 10 miles around the Infected Place, is imposed, and all stock within 2 miles of the outbreak are inspected and kept under observation. No movement of animals out of the area is allowed and movements into and within the area are controlled by licence, only necessary movements being permitted.

Infected Area restrictions include the prohibition of hunting, point-to-point meetings, coursing, closure of certain footpaths, restrictions on markets, also curtailment of troop manoeuvres and of the visits of personnel to farms. Veterinary surgeons practising in Infected Areas are immediately informed of outbreaks as are officers in charge of artificial insemination centres. A local foot-and-mouth centre is set up from which are undertaken all the activities necessary for dealing with the outbreak.

In single uncomplicated outbreaks the Infected Area is reduced to a radius of about 5 miles 14 days after the confirmation of the outbreak, and is completely withdrawn after a further week. The restrictions on the infected farm are maintained, however, for about 6 weeks from the completion of slaughter.

Artificial insemination is discontinued in the whole of the Infected Area for 7 days following an outbreak ; thereafter it can be resumed up to within 5 miles of the infected farm with semen from a centre outside the area or from any sub-centre and, after 14 days, to within 2 miles, except on farms which are under special restrictions because of some connexion with the outbreak. This is the procedure which is followed in single uncomplicated outbreaks, but if there are more than one it may have to be varied.

In outbreaks where there is reason to believe that there may be widespread distribution of disease as a result, for example, of the movement of infected animals through a market, Controlled Area restrictions may be imposed around the Infected Area. The Controlled Area is far larger than the Infected Area and its purpose is to hold animals until they have been traced, examined and placed under restrictions. On occasions, when large markets are implicated, these tracings may run into several thousands and cover a very large part of the country. The Controlled Area restrictions are usually withdrawn when this work has been accomplished or, if they have been imposed for other reasons as, for example, when there is evidence of a widespread invasion of disease from the Continent, when the threat appears to have diminished.

Reasons for the Stamping Out or Slaughter Policy

These are generally well known to veterinary surgeons and it is not proposed to do more than refer briefly to some of the reasons behind the policy. For long periods this country is completely free from foot-and-mouth disease and it is only when infection is introduced from abroad that outbreaks occur. The aim of the slaughter policy is to wipe out these centres of infection as soon as they appear, for this very infectious disease cannot be contained under the conditions prevailing on the ordinary farm. If it were allowed to establish itself it would soon become widespread in our highly susceptible and densely populated livestock and would eventually become uncontrollable and endemic.

Prompt action is necessary, therefore, to destroy the infected animals—which are active manufacturers of virus—and those which have been in contact with them, to prevent this happening. This is what the slaughter policy is designed to do and has done despite many severe tests during the past 60 to 70 years. The policy is not peculiar to Great Britain

but is used in most progressive countries and, probably, would be adopted by many others if they were more favourably situated geographically.

The success of the slaughter policy depends to a large extent on co-operation and speed. Co-operation from the farmers in reporting suspected disease with the utmost promptitude, and speed in diagnosing and wiping out centres of infection as soon as they appear. Delay in either of these operations, particularly delay in reporting, may cause great damage, and, furthermore, is a contravention of the foot-and-mouth disease Orders. If cases are reported promptly by the farmer or veterinary surgeon there is generally very little spread, and our worst epidemics have usually been associated with delay in reporting the disease.

Vaccination has often been advocated as an alternative to the slaughter policy, or a combination of vaccination and slaughter. Careful thought has been given to this matter and the considered opinion is that, under the conditions existing in this country, there is no satisfactory alternative to the slaughter policy at present. In reaching this decision account has been taken of the multiplicity of virus strains and variants of these strains all requiring their own vaccines, the comparatively short period of immunity conferred by vaccines, necessitating re-vaccination twice or thrice annually to maintain an immune livestock population, the limitations of polyvalent vaccines, doubts about the effectiveness of vaccination of pigs, sheep and young animals, and, above all, the labour involved in universal vaccination and the cost which it is estimated would exceed by many times the amount spent annually on the slaughter policy.

As regards the efficacy of vaccination in the vicinity of an outbreak to prevent spread to neighbouring farms there are serious drawbacks to this procedure. Firstly, it is necessary to type the virus to ensure that the appropriate vaccine is used, and, secondly, as it takes about 14 days for the vaccine to confer a satisfactory immunity, the probability is that, if further spread occurs, the vaccinated animals would be infected before their immunity was established. In any case, where there has been prompt reporting, the number of primary outbreaks from which secondary spread takes place is comparatively small. In the view of the Departmental Committee on Foot-and-Mouth Disease (1952/54) "ring" vaccination would not in normal times make any contribution to checking the disease which would justify the expense and effort it would entail.

Occasionally owners of valuable herds have sought permission to vaccinate but their requests have not been granted. The main reason is that if vaccinated animals were exposed to infection when their immunity was on the wane, or before a maximum immunity was set up, they might contract the disease in a mild or masked form without showing visible evidence of it and they could become a source of infection to other animals.

Vaccination, without doubt, is of considerable value as a method of reducing the incidence of the disease in those countries where it is endemic, but it is no substitute for the slaughter policy under the conditions prevailing in this country.

The Importance of the Disease to the Livestock Industry and the Veterinary Profession

The Departmental Committee appointed by the Minister in 1924 to consider the outbreak of foot-and-mouth disease had this to say about the disease :—

> "The real trouble with foot-and-mouth disease is not its deadliness but its extraordinary infectiveness and we do not have only to consider its effect upon the animals attacked but on the flocks and herds of the United Kingdom as a whole. If once foot-and-mouth disease were allowed to become endemic the total losses which would be suffered by traders and dairy farmers would be gigantic and all stockholders would be faced with a constant and recurring menace to say nothing of the effect on our export of pedigree stock which is of importance to the meat and wool trade of the world."

The Committee appointed in 1952/54, that is 30 years later, endorsed this view and stated, furthermore, that the disease would rapidly become endemic in any country which failed to take energetic and rigorous steps to prevent it, and, if this were to happen in this country, the result would be a national calamity.

These are the views expressed by two Departmental Committees on the importance of this disease both to the country and the livestock industry.

The veterinary profession, for its part, is charged with the responsibility of detecting and eradicating the disease whenever it appears in this country, and, moreover, is often judged by the skill and alacrity with which it performs these functions. Surely no more vital role could be assigned to it.

IN PARLIAMENT

Charollais Bulls

On May 4th, SIR A. BOSSOM asked the Minister of Agriculture, Fisheries and Food what progress has been made in regard to convening the meeting of interested parties on the question of experimental importation of Charollais bulls.

MR. JOHN HARE replied that he was considering the comments of the various organisations that had been consulted on this question and he hoped to be able to make a further statement soon.

Rabbits

SIR A. BOSSOM also asked the Minister of Agriculture, Fisheries and Food, in view of the increase in rabbit pests, whether he will make a further statement on the progress being made by his Department in encouraging the formation of rabbit clearance societies.

MR. JOHN HARE said that on March 26th there were 135 rabbit clearance societies in England and Wales. Since then a further 45 had been formed bringing the total to 180 societies covering about 3,000,000 acres. In addition, a further 150 societies were in process of formation.

Animal Husbandry: Present and Future*

BY

SIR WILLIAM SLATER

WHEN we see sheep slowly grazing on the hillside or cattle browsing gently in the meadows, we rarely stop to think of the host of complex biochemical mechanisms going on behind fleece and coat—mechanisms which must never fail if the animal is to remain healthy and so fill its allotted place in the farming system. Since the aim of animal husbandry must be to provide conditions under which these mechanisms can function normally, I make no apology for devoting a large part of this lecture to the consideration of the nature of these biochemical processes.

Influence of Rumen

The food which any animal eats must first be broken down by a sequence of digestive processes into substances which can pass through the gut wall into the blood stream. In the ruminant the bacterial activity in the rumen not only enables the animal to break down and utilise plant tissues, which are untouched in the digestive tract of the single-stomached animals, but the bacteria also synthesise from the simpler products of digestion complex compounds not originally present in the food. This greatly complicates the study of nutrition, and particularly of metabolic disorders in sheep and cattle. If we feed a rat on a diet known to be deficient in some constituent—for example a particular essential amino-acid—we can be sure that the animal will suffer from a lack of this substance. With a ruminant this can never be assumed ; the missing substance may well be synthesised by the flora of the rumen and made available to the animal. Let me give one simple example. It is possible to feed a diet deficient in protein to a ruminant without ill-effect, if urea or even an ammonium salt is given in place of the protein ; but to do so successfully the animal must be receiving in its ration at the same time a larger amount of easily digestible carbohydrate—sugar or starch—than would otherwise be wanted ; this carbohydrate is needed to provide the energy for the increased rumen activity involved in converting the urea into protein. Although this difference between the ruminant and the non-ruminant is generally well recognised, its consequences are often overlooked in considering the sequence of events resulting in a metabolic disorder. We know so much of the deficiency disorders in the non-ruminants that we are tempted to apply this knowledge to the ruminants without making the reservations necessary in such an extrapolation. I shall return to this function of the rumen when I am considering some of the metabolic disorders in sheep.

The Body's Transport System

The products of digestion pass through the gut wall into the body's transport system—the circulating blood. The more we learn of this transport system, how it works and how it is regulated, the more wonderful it appears ; the more remarkable that it fails so rarely to deliver the right goods in the right quantities to the right place at the right time. It makes the most complicated system of automation in industry seem childish.

Its most important, but in some ways its least complicated, function is to carry the oxygen required for combustion to every part of the body and to remove the resulting carbon dioxide : important in that a failure in the oxygen supply means immediate death, less complex in that it is a continuous process which is directly self-regulating through the carbon dioxide concentration. The removal of waste or excess products from the different biochemical processes to the kidneys for excretion is the next most important task the blood stream performs. Again, like the transport of oxygen, the process must be continuous, but its breakdown is not so immediately fatal. An animal cannot, however, live for long if the kidneys fail to remove from the blood the accumulating waste products. The control of this process of elimination is more complex than that of respiration, in so much as we are dealing here not with one substance but with many. A study of what is being excreted through the kidneys in the urine is an important guide to abnormalities in the blood.

Apart from these two functions, the blood stream carries from the gut wall the materials required for energy production and for building and repairing the cell structure to every part of the body. These substances may be wanted in large amounts, like the carbohydrates for the energy supply and the proteins for building into the body, or in amounts so small, in the case of the vitamins and organo-metallic compounds, as to be almost insignificant ; yet we all know these traces of highly complex substances are absolutely essential if the body is to continue to function normally.

The control of the level of the many constituents of the blood rests primarily on the nervous system and on the endocrine glands. These are closely interrelated in that not only do they profoundly influence each other, but they jointly control the functioning of other cells of the body by the nervous impulses put out by the one and the hormones secreted by the other. Thus if an animal is frightened or excited the nervous system affects the adrenal glands and adrenalin is secreted into the blood stream ; this in turn causes the liver to break down glycogen to glucose and the level of sugar to rise in the blood. When the cause of the alarm has passed, the adrenalin level falls, the excess sugar is converted into glycogen and the animal returns to normal. Again, the rhythm of the sexual cycle and the complicated processes of reproduction and lactation are controlled by glandular secretions into the blood stream.

The study of the changing levels of these many different blood constituents offers to us the best

* The second of two Share-Jones lectures delivered in the University College of North Wales, Pangor. The first lecture was published in THE VETERINARY RECORD last week.

chance of following and understanding changes in the metabolic processes going on within an animal and of correlating these with externally observed symptoms.

Levels of Blood Constituents

With all the many biochemical processes going on in the animal body, and the complicated controls involved, it may appear surprising that obvious abnormalities do not occur more frequently than they do. The reason lies in the relatively wide fluctuations in body constituents and body function which are possible without an animal becoming abnormal in any way or appearing ill, and in the regulator mechanisms which are immediately and automatically called into operation when there is danger of the limits of normal fluctuation being exceeded.

When, however, we come to consider what are the limits of fluctuation in any blood constituent which may be fixed as normal for reference purposes, we find ourselves in difficulty owing to the differences between individual animals. In the last lecture I spoke of visible genetic differences resting on primary biochemical differences, these being in turn controlled by the genetic structure of the cell nucleus. Every animal with its individual genetic make-up has, equally, its own individual biochemical organisation. The broad general biochemical picture is, of course, the same in all animals, and particularly in animals of the same species, but the level of activity, the limits of fluctuation and the end point of the same reactions vary.

We are apt to take for granted that certain easily measurable characteristics are fixed for all animals of one species. Thus in man we speak of 98.4° F. as the normal temperature of the body. This is no more than a reference point from which most individuals should not vary markedly in health. There are, however, quite a number of fit healthy people whose temperature is consistently two degrees, or even more, below the figure we have been taught to expect. In other words, the biochemical thermostat of their bodies is set lower than that of most of the population. The same is true of pulse rate, some of us have a faster heart beat than others, but there is no reason to assume that the one rate, provided it is consistent under identical conditions, indicates a less healthy condition than the other. Long distance runners frequently have a very slow heart rate. This kind of variation applies to all the measurements we can make on the animal body.

The use of blood composition in the study of disease must therefore be preceded by an extensive study of the levels found in normal healthy animals. We must have a base line with which we can compare the level found in the sick animal and to which we can follow the level gradually returning as the animal recovers.

Variations in these Levels

Not only does every animal have its own individual biochemical make-up; in addition these biochemical characteristics are not constant for any one animal, varying with nutrition, environment, age and the period in the sexual cycle.

The changes in any particular constituent of the blood of any one animal may follow a regular pattern depending on a physiological rhythm, such as in the sexual cycle, or they may be random in character due to the immediate influence of external conditions. If, however, the animal is healthy and is not subject to extraordinary stress, which produces conditions similar to those in disease, the variations in the level of the blood constituents are kept between fairly well defined limits by the physiological control mechanisms of the body. It is possible therefore to determine for any healthy individual, if enough measurements can be made under different conditions, the range of variations for the constituents of the blood.

If, however, we determine the range for a number of apparently similar animals we find, as is to be expected from their differing genetic make-up marked differences in the results. In one animal the maximum value of the range may be higher, in another the minimum lower; in one the spread of the results may be wide, in another narrow. We have therefore to contend not only with variations within any one animal, but also with differences in the amplitude and form of these variations from one animal to another.

We can, however, by taking enough measurements on many animals build up a composite picture, showing the range of variation in health for each blood constituent. The range for most of the animals will fall within a central band, but some of the minima will be below this and some of the maxima above. It may be useful to explain this with a somewhat crude analogy. When a potter throws by hand a number of similar vessels they all differ slightly in shape. If we now take photographs of all the vessels and superimpose the transparent prints one on the other, we shall get the dark outline of a vessel where all the separate shapes overlap. Around this dark central shape will be a blurred area growing gradually fainter until it merges into the background of the print. This represents the variations outside the average shape, which grow fewer as they diverge more from what the potter had in mind. Yet none of these vessels will appear abnormal; the variations would be too small.

We can in the same way by making many observations equip ourselves with a series of values for the constituents of blood which can be regarded as normal, since a large majority of the determinations fall within the range. Albritton, who has published tables of these values, takes the spread covered by 95 per cent. of the results as representing the normal range. The remaining 5 per cent. represent the blurred fringe around the image of our potter's vessel. Although for a time I want to ignore these values lying outside the normal range as defined by Albritton, I shall have to return to them later; they are the possible explanation of some of the most puzzling problems in animal husbandry.

Growing Importance of Metabolic Diseases

If a farming audience had been asked in, say,

1920 to write down the most serious diseases of sheep and cattle in order of the damage they caused and a similar group of farmers to say what were the diseases which worried them most to-day, the lists would show marked differences. There would be many changes in the order of importance; some diseases rated high in 1920 would now hardly find a place on the list, whilst conditions unrecognised in 1920 would now be near the top. But it is in the pattern of the change that we should be most interested. The diseases which have decreased in importance will be seen to be those caused by the invasion of the body by a pathogenic organism which produces a recognisable diseased condition: those which have increased in importance will be of two types; the one where an organism can be present in the body for an indefinite period with no ill-effect, until suddenly some change in metabolism triggers off a rapid increase with a corresponding production of toxic effects; and the other, where no pathogenic organism can be found but some change in environment, producing undue stress, results in a gross metabolic disturbance.

Two factors have led to this change in pattern. The first is the great increase in our knowledge of bacteriology and virology and of the different means of immunisation, compared with the relatively slow advance in the study of the physiology and biochemistry of farm animals. The second is the general trend towards higher farming standards. As we expect to get greater productivity from our animals by better breeding, management and nutrition, we must at the same time remember that we are increasing the strain on their metabolism. We may easily, if we are not careful, push a biochemical process beyond the point where the self-balancing mechanism operates; once over the edge only by prompt and sometimes drastic action can it be pulled back.

The study of the incidence of these diseases of metabolic origin immediately raises the problem of why in a flock or herd only a few animals may be affected, although all have been bred and raised under the same conditions and so far as is known subjected to the same physiological strains. The explanation I believe lies in the biochemical variability—the blurred fringes around our potter's vessel.

If we consider the many different biochemical and physiological measurements—some closely interrelated, others quite independent—which can be made on an animal, it would be surprising if for some at least the values did not fall outside the normal range, and for these the animal may be said to be abnormal.

Influence of Genetic Variations

Genetic variations of this type may not affect an animal adversely at any time in its life. It may never be subjected to those stresses which would change a minor controlled abnormality into one which is uncontrolled and catastrophic. But it is the animals in a flock or herd which have these variations from the normal in their biochemical and physiological processes to a major degree which are most likely to succumb to metabolic diseases. This point can be illustrated by studies which have been made of such a condition in man. Insulin, which controls the carbohydrate metabolism, is secreted by the Islets of Langerhans in the pancreas. It is well established that the amount of islet tissue may vary tenfold as between one man and another, and its capacity to produce insulin to the same extent. Men with values at the low end of this range are liable to develop diabetes, if at some time in life they are subject to a condition which tends to reduce the production of insulin, for example a loss of some of the islet tissue. On the other hand, those with a high insulin level are liable to suffer from hyperinsulinism. Neither the man with high level or the one with the low may ever suffer in health if they are not subjected to some unusual and abnormal stress.

Or put in another way, this simply means that in some animals the control mechanism which stops a reaction from getting out of hand acts sooner, either in time or in quantity, than in others. As with a man driving a motor car, the quicker his reaction time the less likely is he to have an accident; but the man with a slow reaction time may never have trouble because through care or luck the call for quick action never arises. If, however, 100 men with unknown and varying reaction time had to drive along a dangerous road at high speed some would crash; the more dangerous the road and the higher the speed the more crashes there would be as the reaction time of more and more men failed to meet the need.

Under modern conditions the stockman must think of the animals under his care in this way. He must think less of the possibilities of infection—provided he can take care of these by immunisation—and more of the stress which he is putting on the animal's metabolism when he intensifies production. Because some animals in a flock or herd escape infection, or are already immune to it, the stockman has come to accept the idea that a disease will not strike every animal, but he is, rightly, apt to consider that this is just the luck of distribution, or a clear-cut difference between immune and non-immune animals. He watches for the first signs of disease and then takes action, if he has not already done so, against a foe invading from without. With metabolic disorders he must learn to anticipate the effect of each change of environment and nutrition in the light of his knowledge of the history of his animals, and their present productive state, that is, for example, whether they are pregnant or lactating heavily. With the concept in mind that some animals—those with metabolic limits just outside the normal range—will be more sensitive to the change than others, he must watch for the earliest signs of trouble in any one animal. Once this is recognised he should try to ease the stress on the rest of the flock, because if it continues other animals will go down one by one as their tolerance limits are overstepped.

This is no plea for turning back on the road we are following to higher output from the land, but it is a plea to go cautiously and with an understanding of what we are doing. Highly skilled stockmen

under carefully controlled conditions can, for example, produce very high milk yields without harm to their animals, but to aim at producing these results on all farms would be folly. Even with all the care the cows have in the successful high yielding herds, they not infrequently run into metabolic troubles. Gradually we are finding the solution to these, but an outbreak of such a metabolic disturbance can cause losses in the most productive herds, which will rapidly wipe out any profit arising from the higher yields. The same general principles apply in varying degrees to all types of stock. With sheep the stresses are more difficult to control, arising as they do more from changes in natural conditions supervening on the planned management.

Some Diseases of Metabolic Origin

At this point I should like to turn to the more detailed consideration of some of the diseases of metabolic origin, particularly in sheep, and to the nature of the stresses which cause them. One condition which is claimed to be increasing is that known as "Lambing Sickness" or "Milk Fever." It is associated with good pasture and high milk production and is due to the fall of the calcium level in the blood beyond the limits required to maintain health. The first idea which must occur to anyone considering these facts is that the pasture being lush may be deficient in calcium and hence the ewe, requiring, as she does, large amounts of calcium for her milk, is thrown out of balance. Examination of the pasture, however, does not show any shortage of calcium which can be correlated with this disease. The cause lies in a failure of the mechanism which controls the level of calcium in the blood. How exactly this has failed we do not know. What we do know is that in the normal healthy ewe calcium is mobilised from her own skeleton to make up any deficiency in intake during lactation and that the disease responds rapidly to subcutaneous injection of various calcium preparations.

We can well ask ourselves therefore whether this may not be one instance where the animal under ordinary conditions has a blood calcium level below that accepted as the normal range; that is where genetically the control mechanism is set low and hence much nearer the danger level. So far as I am aware this has never been tested. If in the same animal the endocrine control of milk secretion is such as to produce a high yield of milk, the genetic make-up may be right for the animal to go down with the disease. Even if we accept this predisposition to the disease, we still have not explained what suddenly turns an animal which, though having some abnormal biochemical characteristics, is healthy into one which is so sick that it may die within a few hours. Again we can speculate on the cause. It might, for example, be that at a particular stage of growth reached by one of the grasses or legumes in the pasture a substance is produced, hormonal in character—we know such substances are present—which suddenly stimulates the milk production to an extent which depletes the calcium level more rapidly than it can be replaced. If it is already normally at a low level, it may fall below that at which the whole metabolism is disrupted and the animal becomes seriously ill. If this speculation is true it would explain the spectacular effect of injecting calcium, in that this would raise the level long enough for the normal mechanism to take over again. Such speculations do not solve the problem, but they do give us valuable starting points for further investigations.

One question arises if we accept that there is possibly a genetic tendency in the animals which succumb to the disease. The discovery that the condition can be rectified by the simple subcutaneous injection of calcium must mean that relatively few ewes are now lost through the disease. Those which survive may remain in the flock and produce more lambs. The number of surviving lambs from affected ewes may also be greater and they too may be kept for breeding. When the disease was nearly always fatal, the animals with a genetic tendency to develop it—if it is in fact a correct assumption that there are such animals—would tend to be eliminated from a flock; with modern treatment they would be retained and the disease would persist and might under some circumstances of inheritance become more frequent. It might therefore be wise, until the assumption of inherited tendency is proved to be false, not to retain for breeding in the flock the animals which have recovered from the disease or their progeny.

Another metabolic disease associated with rich young pasture is "Grass Staggers." In this case the cause of the observed symptoms is a low level of magnesium in the blood stream and it can be successfully prevented by feeding compounds such as magnesium oxide or magnesite. There is no obvious explanation for the disease; once again we can only say that the control mechanism seems to have failed in some of the sheep.

I should now like to consider certain other metabolic diseases where genetic abnormalities do not seem to have any part. One of the most interesting of these is known as "Pine" in sheep. This disease illustrates well the important part which the rumen flora play in nutrition by their ability to synthesise complex organic compounds. The condition has been known for over a century. It was described as occurring on Dartmoor at the end of the 18th century. The treatment prescribed was either to send the affected lambs to other pastures, or to bring soil from areas where the disease did not occur and use it as a drench. It was not, however, until after the first World War, when the disease became serious in Australia and New Zealand, that it was fully studied. The symptoms were such as to suggest an anaemia and drenching with crude iron sulphate was found to protect the lambs. When, however, in the course of the experimental work a purer iron preparation was used, it failed to prevent the condition occurring. The next obvious step was to try the impurities which had been present in the original iron sulphate and it was found that the small trace of cobalt present was responsible for the curative action. The treatment—the addition of a few parts per million of cobalt to the food, or the spray-

ing of the pastures with 2 lb. per acre of cobalt sulphate—was now established, but the part which the cobalt played was not known. Another chance observation gave the clue to this. It was found that the injection of cobalt into the blood stream had no effect; it must be administered by mouth. The only conclusion to be drawn was either that the cobalt was essential for the activity of the rumen flora, or the bacteria in the rumen synthesised a complex cobalt compound essential for the well-being of the sheep, or that the cobalt was required for both. At this stage the possible solution was provided by work in human medicine on the B vitamin complex. It was found that one member of this group B_{12}, which is essential to the animal's well-being and without which serious disorders of the blood occur, is a complex organic compound containing a small amount of cobalt. If this vitamin injected into the blood stream protected the lamb, then it could be regarded as proof that it was synthesised by the rumen flora. Experiments showed this to be true. It has also been shown that cobalt is an essential constituent of the rumen bacteria.

I have discussed "Pine" at some length because it illustrates so well the important part played by the rumen and the need to keep this constantly in mind in studying conditions of malnutrition.

Effect of Metabolism on Invading Organism

In considering metabolic diseases it is impossible not to refer to those conditions which result from the interaction between an organism present in normal healthy animals and a change in nutrition or environment, which would otherwise cause no ill effect. Perhaps the best understood example of this type of disease is that known as "Pulpy Kidney" or "Enterotoxaemia." In this disease healthy rapidly growing lambs—usually the most forward in the flock—die suddenly at 3 to 8 weeks old. It is due to a toxin produced by an anaerobic organism which is normally present in the intestine, but in harmless numbers. A sudden change to good pasture, with an accompanying rise in the ewes' milk, creates in the intestine conditions for the rapid multiplication of the organism, and the production of the toxin in dangerous and often lethal amounts. Fortunately the lambs can be protected by a vaccine giving active immunity, or by a serum which gives rapid but not lasting protection.

Here we have a condition where the form of the stress is well established and its consequences known. There is no question of an hereditary tendency in the lamb—other than the good milking quality of its dam—nor is the upsetting of the balance of a biochemical mechanism involved. The treatment too can follow the lines which have proved successful in so many diseases, namely the preparation of a vaccine.

Johne's disease, which is most serious in cattle but also occurs in sheep, is similar in character. The organism which causes the disease can be found in many quite healthy cattle which never develop the disease. Yet any one of these animals may suddenly, and for no apparent reason, become acutely ill with all the characteristic symptoms of the disease. Here we do not know the nature of the stress which causes the sudden change, nor have we yet been able to protect cattle by immunisation, although promising work is in progress.

Parasitic Diseases

No discussion of the problems of animal husbandry would be complete without some consideration of the internal parasites which cause such heavy losses in our livestock. In fact one of these, the "Liver Fluke," is at the present time rated as one of the most serious troubles in North Wales.

The simplest type of parasite, that is the one with the least complicated life cycle, is the group comprising the stomach and bowel worms, which do not leave the intestines of the animal for another site in the body. The eggs of these worms are voided by the infected animal on to the pasture, where under favourable moist warm conditions they hatch into larvae which, after the necessary development, make their way up the stems of the grasses, and particularly the clovers, and are eaten by the animal. In a short time they develop into adult worms and so continue the cycle. From this description it might be thought that animals grazing any pasture intensively would become increasingly infested with parasites. There are, however, two factors which operate against this continuous increase. The first is that the larvae have to have the right conditions for survival. Cold dry weather will result in many failing to survive. If the animals are moved from pasture before the voided eggs have had time to hatch and develop into larvae, that is within 4 days, and not returned to it until most of the larvae have died, the infestation can be controlled. The problems involved in this control by management are the obvious one of the availability of fresh pastures, and the less simple one of estimating the length of time which should elapse before the animals return to the original field. The time interval depends on the weather, if it is cold and dry 4 weeks may be enough, but if it is warm and moist twice the time will be required. Unfortunately the weather which favours the larvae also favours the growth of pastures, so that when the farmer can risk moving his animals at the shorter time the previously infected pasture may not be ready for further grazing; whilst when he has to wait for the longer period he may have to watch the infected pasture getting past its best grazing period, before he can use it again.

The second controlling factor is that the animals develop an immunity against parasitic worms, just as they do against bacteria or other invading foreign bodies. Although this has been known for some time, many of the details of the immune reaction have still to be worked out. We know, for example, that when a sheep has been heavily infected the antibodies conferring the immunity rise in the blood, reaching a maximum after a relatively short time, and then decline; and that whilst the immunity is high a fresh infection of larvae fail to develop and produce eggs. Until we know more about this immune reaction, we cannot make positive use of it in protecting our flocks.

One very interesting development, however, has

been made in the treatment of Husk, Parasitic Bronchitis, or Lung Worm in calves. The larvae of this parasite works its way from the intestine *via* the lymph glands to develop into the adult worms which infest the bronchii causing coughing, difficulty in breathing and in bad cases, death. It was known that animals which had recovered from an attack were largely immune; larvae fed to them failed to develop. It was further found that serum from an animal infested with the worms when injected into another calf gave a considerable degree of immunity. This resistance to the disease—passive immunity—however only lasted for a short time and involved maintaining large numbers of infected animals to produce the serum. The next step was to try to find a weakened strain of larvae which, whilst not developing within the animal, would confer active immunity of a lasting character. Larvae were submitted to different levels of radiation until one was found which so weakened the larvae that they travelled only as far as the lymph glands, where they failed to develop and did no harm. Calves fed these larvae have been shown to be highly immune. Now we are left with the problem of obtaining larvae on a sufficiently large scale, so that they may be irradiated and used in immunising calves. If this is achieved it should add Husk to the other diseases over which we have gained control.

The Menace of Liver Fluke

Unfortunately Liver Fluke, which worries you so much in North Wales, is not an easy disease to control. Although I expect most of the audience already know the life cycle of this parasite, I will give it briefly. It is perhaps simplest to begin where the eggs are voided by an infected animal. These hatch within 3 or 4 days into a form which is very mobile in water. This small creature must within about 14 days find a particular freshwater snail, which is its secondary host. As these snails live in shallow stagnant water and muddy places—ditches and the edges of puddles—the fluke stands no chance of survival if its eggs do not fall near such a place. The young flukes which enter a snail develop and multiply over a period of 6 or 7 weeks, at the end of which time they emerge and swim about independently seeking an anchorage. When it finds one the fluke attaches itself by secreting a cement-like material around itself. In this form it has been shown to survive up to 12 months, although most of the encysted fluke die within 2 or 3 months. If the anchorage of the cyst is a blade of grass, the grazing animal will take it in with its food. The cysts on reaching the intestine become active and the fluke, breaking out, pierces the gut wall and enters the abdominal cavity. From there it finds its way to the liver, where it causes most serious damage. It burrows its way through to the smaller bile ducts and to the larger ducts and the gall bladder; there it lays its eggs which find their way down the bile ducts to the intestines and so to the pasture.

There are two ways in which the flukes can be attacked; by drugs whilst they are invading the liver and gall ducts, or by eliminating the snail—the intermediate host. The latter can be done either by draining and cleaning the pastures so that the snails have nowhere to breed, or by destroying them by applying dressings of copper sulphate to the pastures. Unfortunately the drugs are not fully effective in doses small enough to be safe for the animals. They kill the adult flukes, but are not effective against the thousands of immature flukes which invade the liver in acute cases. If pastures can be drained so that conditions are unsuitable for the development of the snails, it is possible to eliminate the disease, but particularly on heavy land this may be impracticable. Failing this the dressing of the pastures with $\frac{1}{4}$ cwt. copper sulphate to the acre will keep the snails in check, but it has not proved entirely effective. To be so the farmer must be able to recognise the snails and to locate the areas where they are breeding; he can then dress these areas in the early summer and repeat the dressing whenever he sees the snail reappearing.

This in total gives the farmer a difficult task to control this parasite, if his land is already infested and is not easily drained. One further difficulty is that the fluke does not have in general so serious an effect in cattle as in sheep, and hence cattle may act as carriers where there is mixed grazing without showing any obvious signs of infection.

What can we hope for in the future? My own view is that we are unlikely to find an effective drug which will kill the fluke at all stages; particularly when it is still immature and is working its way through the liver and causing major damage without at the same time poisoning the animal. It is not impossible that someone may do so but I do not think the prospect very hopeful. There may be a better chance of finding some chemical more lethal to the snails, which would make the spraying of pastures easier. The fact that a chronic condition exists in which an animal will carry a low count of mature flukes for several weeks and then recover may suggest a slow building up of immunity, although as far as I am aware this has not been shown. If this proved to be so then it might be possible to attack fluke on the same lines as husk, although the task would undoubtedly be very difficult. I am sorry to end the discussion of Liver Fluke on this unhelpful note, but I should be wrong to suggest that any easy solution is in sight.

The Pig-keeper's Problems

So far I have not mentioned pigs, not because I regard them as less important but because pig husbandry differs markedly from that of sheep and cattle. Here we have no problem of rumen metabolism; the pig by and large depends on what it is given for a complete diet. Hence we may extrapolate with much more confidence, if still with caution, from experiments on laboratory animals. Again, the pig spends most, if not all, its life in a building and is given a ration which is under the stockman's control. The problems posed by the pig-keeper are quite different from those of the shepherd and the cattleman. The pig-keeper is seriously concerned about the right type of building, both for farrowing and

for fattening. How far will it pay to provide additional insulation? What is the optimum temperature for a piggery, and how will variation from the temperature for either short or long periods affect his results? What is the right humidity at which to aim? How can the labour of feeding and cleaning be cut to a minimum? These are the questions he asks.

Of equal importance to the design of the piggery is the proper balance of the diet. The major factor in this is the quality of the protein used. There is only about half the quantity of white fish meal available which would be required, if this alone supplied the animal protein supplement in pig and poultry food. Hence it is necessary to use many other materials to make up the balance. The amount of protein in any material is calculated on the estimation of the nitrogen it contains. For the high quality fish meals and for other well manufactured products this corresponds with reasonable approximation to the biological value, that is the value determined by actual feeding experiments. If, however, the materials have been overheated, or otherwise maltreated, in manufacture, some of the essential proteins are rendered useless and the nitrogen value no longer corresponds with the biological. If damaged materials of this kind are incorporated in a ration for pigs, their growth will be retarded and the other materials making up the rest of the diet partly wasted. The feeding stuffs manufacturers are most anxious to avoid incorporating any such materials in their rations, but with the rapid turnover of supplies they must maintain it is quite impossible to do feeding tests on every batch. We are therefore concerned to find a rapid laboratory test, either chemical or microbiological, which will give a reasonably accurate estimate of the biological value. Co-operative experimental work is now in progress in which scientists from the feeding stuffs manufacturing firms are co-operating with those from the universities and the research institutes.

Pig diseases, because of the control of diet and environment, tend to be more concerned with invading pathogenic organisms than with metabolic disturbances. Moreover, the continuous close confinement of the pigs within a limited space makes the spread of infection easier. There are many important problems in clinical pathology for which the pig-keeper requires a solution, but most of them lie in the fields of bacteriology and virology.

There are also a number of more or less artificially created problems, such as those involved in finding the correct diet for the early weaning of suckling pigs, which are calling for special attention.

The pig-keeper is vitally concerned with the carcase he produces, because on its quality depends the price he receives. The type of carcase depends on the interaction between the pig's genetic make-up and the type and quantity of food it receives. The aim is to produce the top grade carcase as quickly as possible and with the minimum cost in food. These are to a large degree opposing factors and hence it is impossible to achieve an optimum value for each; what we have to do is to find the best point at which to aim with our existing knowledge and the prevailing price structure.

Poultry—a Growing Industry

I have left little time in which to speak of the poultry-keeper's problems. This industry has within 30 years changed from being a sideline on a general farm—often the perquisite of the farmer's wife—to a large scale commercial production. Not only has the production of eggs greatly increased, but since the second world war we are rapidly moving in this country towards the position in the U.S.A., where poultry meat is cheaper than beef or mutton. This change to intensive large scale indoor production in batteries and deep litter houses has created a wide range of problems, similar in many ways to those of the pig-keeper. There is, however, one major difference; we know far less about the basic physiology and biochemistry of the domestic fowl than about the pig. The fowl, although it is easily kept, has never been a popular animal in the medical laboratory because it differs so markedly from man and we cannot extrapolate from the other laboratory animals to the fowl as we can for the pig. On the other hand, owing to its rapid rate of reproduction and the relative ease with which it can be kept, it has been extensively studied by the geneticists. As a result, the breeding of poultry has advanced more on scientific lines than that of any other class of stock. In the United States the large hatcheries are employing numbers of highly trained geneticists and the same trend is developing in this country.

The poultry-keeper has the same problem as the pig-keeper with regard to protein quality, and the two can be solved together. He has also similar problems concerned with housing, but these require special independent investigation. There are many problems in nutrition—particularly in the laying hen, where the turnover in protein and mineral salts is so large in relation to bodyweight—which are awaiting solution. Finally, and most important of all, is the prevention and cure of the infectious diseases which run so quickly through birds kept in close confinement and have disastrous effects on the poultry-keeper's profits.

Conclusion

The lecture has perforce had to range over many subjects and I am afraid it has been discursive and to some degree fragmentary. The subject is so vast that to cover it with any degree of completeness would require a long course of lectures. I have, however, tried to link what I have said to what are in my view the most important general problems of animal husbandry. The first is the need for a much fuller knowledge of the physiology and biochemistry of farm animals, if we are to succeed in solving the most urgent questions posed by the farmers. We are so often groping and guessing our way towards an answer, because we do not know how the organism works. Trial and error may produce chance solutions, but the results come slowly and often there is no answer at all. In an attempt to fill these gaps my Council is intensifying the basic physiological and biochemical research work it supports in its own institutes and in the universities. At

(*Concluded at foot of col.* 1 *overleaf*)

A Report on the Use of Promazine Hydrochloride in Equine Practice

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SUMMARY.—Promazine hydrochloride was injected intravenously or intramuscularly into 37 horses. It was used in cases encountered in practice when sedation or restraint was required. The results appear to indicate that it has useful applications in equine practice.*

The Drug

Promazine hydrochloride is a phenothiazine derivative with the chemical composition 10 (3'-dimethylamino-n-propyl) phenothiazine hydrochloride. Although apparently very similar chemically to other phenothiazine derivatives, it has certain definite differences in activity and clinical application.

It is widely used in human psychiatric and anaesthetic practice. Its advantage over other phenothiazine derivatives appear to be its relative lack of untoward side-effects.

Azima and Durost (1957) and Archer (1957) in comparative studies of promazine and chlorpromazine in humans found that they have similar and equal value in their effect on behaviour, but chlorpromazine is more likely to cause a drop in blood pressure due to postural hypotension and accompanied by circulatory collapse. Skin rashes, drowsiness, dizziness, and jaundice are commoner with chlorpromazine and it causes more local irritation on injection than promazine (Atkinson, 1957; Mitchell, 1956; Spira, 1958).

Toxicity

No toxic symptoms were noticed in the horses in which this drug was used.

Schulz (1958) injected a 1,000-lb. horse intravenously with 1.25 g. promazine daily for 15 days. He found no blood dyscrasias or any adverse systemic or local effects.

Seifter *et al.* (1956) report that the LD_{50} (lethal dose for 50 per cent. of animals) for promazine administered by rapid intravenous injections in dogs is 38 mg. per kg.

The toxic symptoms of overdosage in other species appear to be a drop in blood pressure, respiratory depression, and circulatory collapse.

How It was Used

A few months ago it was decided to try using promazine in any cases where sedation prior to manipulation or minor operation was required. It was also to be used prior to induction of general anaesthesia.

In a period of 3 months promazine was used in 37 animals. All were Thoroughbred horses or Hunters.

It was used in the following types of cases and, where necessary, in conjunction with local anaesthesia:—

Castrations in the standing position;
Firings in the standing position;
Suturing wounds;
Clipping difficult horses;
Attention to teeth;
Removing an aborted foetus;
Prior to the application of hobbles for casting;
Colics.

Dosage

Various dosages were tried within the range 0.5 to 2.0 mg. per lb. bodyweight. A considerable individual variation in the dosage required became

* "Sparine," manufactured by John Wyeth & Brother Limited.

Animal Husbandry: Present and Future—*Concluded.*

times we are criticised as being too much concerned with science and too little with agriculture, but it is our considered view that a well planned and systematic attack will in the end produce quicker and better results than a series of sporadic and uncoordinated efforts, which so often in solving one problem create two, each of which is worse than the first.

The second general problem which I hope to have brought out is the increasing complexity of the work of the stockman. Many new techniques and treatments are of little value if they are not put in practice by men with a full understanding of what is involved. Every step we take forward towards higher food production to meet the world's growing population must inevitably increase the stress put on our farm animals. We call for higher milk yields, for more twinning in sheep, for more pounds of pigmeat per pound of food, and more eggs per bird. To achieve these ends—which we must if man is to survive—we increase the rate of reproduction, we feed at higher levels and we house our animals in close confinement. These stresses push the animals nearer to the degree of abnormality which we recognise as pathological and the margin of safety is reduced. Only a well trained and skilled stockman, who understands what is happening in an animal, can be expected so to handle his stock that the safety limit is not exceeded.

I end this lecture therefore, as I did my first, with a plea for greater technical training of the men working on the farms. Whatever we do in the laboratory, on these men must rest the burden of keeping our stock healthy and productive.

apparent. Some highly strung horses needed twice the dose that would have achieved the desired degree of sedation in another animal of similar weight.

At the beginning of these trials relatively small doses were used. In one of the first cases, the removal of an aborted foetus from an excitable Thoroughbred mare, a dose of 600 mg. achieved very little result. In the light of further experience it is felt that this dose should have been larger.

It also appeared that a small dose would often suffice if given to a horse before manipulation was started. However, if the drug had to be used to calm an excited horse in which manipulation had already been attempted without the drug, a much larger dose was needed.

For practical purposes it was found convenient to give an average horse (750 to 1,000 lb. bodyweight) an initial dose of 500 to 750 mg. This was followed in about 15 minutes by a further 250 to 500 mg. if the required effect was not achieved by the first dose.

Administration

Promazine was always given intravenously except in certain colic cases. The dose for the average horse, as presented by the manufacturers, is contained in 10 to 20 c.c. This does not prove too unwieldy.

The Effect of the Drug

To Facilitate Handling

Within about 5 minutes after injection the horse appears to relax. He carries his head and neck lower and stands quietly. His upper eyelid begins to droop. When he moves he tends to drag his feet. There is no evidence of inco-ordination. At this stage the horse can readily be stimulated and is not yet fully under the effects of the drug although he may appear to be. In some individuals there is at this stage an increase in the heart and respiratory rate. Both return to normal in a few minutes. If the drug is being used prior to general anaesthesia with an inhalation anaesthetic, it is very necessary to let the respirations return to normal before administering the anaesthetic.

In 15 to 20 minutes the horse is as completely relaxed as he will become with the dose already given. The head is carried even lower. In the male complete relaxation and drooping of the penis is a marked feature. The horse can still be moved about freely without evidence of inco-ordination or ataxia. The feet will still be dragged. Heart and respiratory rates are rather slower than normal.

The horse is now ready for handling. If at this stage it still shows marked resistance, a further 250 to 500 mg. can be administered. The horse should then be left for another 10 minutes. Any resistance the horse now shows will be at a slower tempo. If he attempts to kick, the initiation and performance of the whole movement will be slower. Horses which normally would violently resist a twitch will accept one at this stage and with it can be fully controlled.

The drug appears to be completely effective for the first 2 hours. The effects wear off gradually during the next 2 or 3 hours.

As a Pre-anaesthetic Agent

Promazine was given prior to anaesthesia with chloroform or deep narcosis with chloral hydrate. It was given in doses similar to those described above. In fact, when it was given, its main purpose was to relax the patient before the application of hobbles for casting. In this rôle it was always most effective.

When used with chloroform it was found possible to reduce the amount of chloroform that would normally have been used by about one third. Also its use appeared to reduce the intensity and duration of the period of excitement associated with induction and recovery from chloroform.

Its use seemed to lengthen slightly the recovery period from chloroform anaesthesia or chloral narcosis.

In Colic Cases

Promazine was used by intravenous and intramuscular injection in a number of selected colic cases. It appeared to give considerable relief.

Discussion and Comparison

Thoroughbred horses, Hunters, and other riding horses vary enormously in their reaction to the strangeness, discomfort, or mild pain of the various interferences to which they have to be subjected. While some are most amenable, many are nervous, stubborn, or vicious and resist with varying degrees of violence. A number of methods of restraint are in common use, such as the twitch, stocks, blinds, sidelines, and hobbles. Particularly with Thoroughbred horses these "mechanical" means of restraint may have an effect opposite to that desired and stimulate the horse to even greater resistance.

The need is often felt for some drug which will render the horse temporarily more docile in order that the minor operation or manipulation may be carried out effectively and with safety for horse and handler.

Morphia or low sedative doses of the barbiturates are not particularly effective and may cause central excitement instead of the desired effect.

Chloral hydrate has been used for many years. When given by stomach tube it takes some time to act. Passing the stomach tube in a vicious horse may not be easy. When given by the intravenous route chloral has many advantages. However, to achieve the required docility it may be necessary to give such a large dose that the horse becomes inco-ordinate. This in itself may interfere with the manipulation for which the drug was given. Large Thoroughbred horses become frightened when they lose control of their limbs and may prove difficult and dangerous to control. Casting may be necessary. Another disadvantage of chloral is the technical difficulty of giving 300 to 500 c.c. of an irritant fluid intravenously to a restive horse. Despite these disadvantages chloral undoubtedly still plays an important rôle as a sedative in horses.

According to Troughton *et al.* (1955) chlorpromazine hydrochloride is effective as a sedative for nervous animals prior to manipulation. They recommend that it should be used intramuscularly. This involves a delay of about 40 minutes and necessitates an accurately computed dose. According to the

(*Concluded at the foot of col. 1 opposite*)

Current Literature

ABSTRACTS

Budgerigars and Poliomyelitis. DANE, D. S., DICK, G. W. A., & DONALDSON, S. N., *et al.* (1959). *Lancet*, March 7th, 1959. 497-8.

This paper describes unsuccessful attempts to infect budgerigars with eleven different poliovirus strains obtained from various sources. Each strain of virus was fed to three birds. On the thirtieth day after virus feeding the birds were inoculated intracerebrally with the same virus with which they had been fed. No virus was found in the faeces of any bird except those of one group in which small traces of virus were found on the first and second day after feeding but at no other time. Seven birds died during the experiment. One died two days after intracerebral inoculation and traces of virus were found in its brain. No virus was isolated from any of the remaining six birds that died.

Search was made for "paralysed" budgerigars and five were received. One had a deformed leg and in the other four there was evidence of a bone fracture.

Follow-up enquiries subsequent to the Belfast outbreak of poliomyelitis in 1957 when there were nearly 200 cases were used to secure information about the presence of budgerigars in the homes. Information was received from ninety-nine poliomyelitis patients, 73 of whom had been paralysed and 26 had suffered from aseptic meningitis. Of the 73 paralysed patients 24 kept budgerigars and of the 26 non-paralysed patients 6 kept budgerigars. Two deaths of budgerigars were reported by the paralysed group of patients, one two months before the patient's admission to hospital and the other two weeks after admission. Two reports of deaths of budgerigars were also received from the non-paralysed group of patients. In the first instance two budgerigars died about the same time as the child became ill; in the second instance the budgerigar died the day after the child entered hospital. It should be noted that the child in the second instance in the non-paralysed group had, a fortnight before his illness, been in contact with a relative who a week after their meeting developed paralytic poliomyelitis. It will be realised that these follow-up enquiries were too late to permit any investigation of the dead birds.

G.F.B.

A Report on the Use of Promazine Hydrochloride in Equine Practice—*Concluded.*

manufacturers there is danger of postural hypotension if it is given intravenously. They recommend minimal dose rates lest overdosing should induce ataxia and panic.

Promazine recommends itself for general use as it appears to have these desirable attributes:—

Effectiveness;
Safety;
Ease of administration;
Quick action;
Freedom from adverse side-effects.

Acknowledgment.—The supply of a quantity of "Sparine" by John Wyeth & Brother Limited, for use in these trials is acknowledged.

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Recent Advances in Anthelmintics. RIEK, R. F. (1958). *Aust. Vet. J.* **34.** 370-81.

In this important paper Riek gives the results of recent work at the Veterinary Parasitology Station, Yeerongpilly, Queensland, on anthelmintic treatment of parasitic helminth infections of calves. These should, he says, be effective against both immature and mature worms of more than one species. Among the anthelmintics tried, Riek found that only toluene, dihydroxyxanthraquinone, and the organic phosphorus compounds Neguvon and Asuntol were promising. Results were based on faecal egg counts done for at least 2 weeks before and after treatment, and on faecal cultures which permitted the identification of the worm larvae. A drug was designated highly effective when it reduced the egg count by 80 to 100 per cent., effective when it reduced it by 60 to 80 per cent., and ineffective when the reduction was less than 60 per cent.

H. McL. Gordon, who opened the discussion which followed, gave a valuable summary of the results of treatment of sheep with the anthelmintics, pointing out, among other considerations, the variable results obtained when the drugs are given to sheep either by injection into the rumen or abomasum, with or without premedication with copper sulphate solution to cause reflex closure of the oesophageal groove, which ensures direct swallowing of the drug into the abomasum. With the same object Riek premedicated calves with 60 ml. of 10 per cent. sodium carbonate. For full details of the results the paper itself must be read, but the main results obtained by Riek in the treatment of calves, and those reported by Gordon in the treatment of sheep, are summarised here for comparison.

Toluene

Riek treated 169 calves, 4 to 12 months old, with toluene at a dose rate of 10 ml. per 100 lb. bodyweight immediately after premedication with 60 ml. of 10 per cent. sodium carbonate. This dose was highly effective against *Haemonchus placei, Bunostomum phlebotomum, Cooperia pectinata, C. punctata, Ostertagia ostertagi,* and "possibly" against

Trichostrongylus axei, but was ineffective against *Oesophagostomum radiatum*. It was more efficient when food and water were withheld from the calves overnight. The drug had no ill-effects, though calves in poor condition sometimes staggered and collapsed 60 minutes after dosing, but recovered spontaneously in 3 to 4 hours. Gordon noted that it was effective against *Haemonchus contortus* in sheep only when it was swallowed into the abomasum; a dose of 10 ml. given into the rumen was not effective.

1:8 dihydroxy anthraquinone

Riek treated 114 calves with doses between 2 and 5 g. per 100 lb. bodyweight. A dose of 2.5 g. per 100 lb. bodyweight was highly effective against *H. placei*, *C. pectinata*, *C. punctata*, and *O. radiatum*, and probably against *O. ostertagi*, but was ineffective against *T. axei* and *B. phlebotomum*, and also against the immature forms of these species. This dose had no ill-effects, but higher doses caused severe purgation in some calves and a dose of 5 g. per 100 lb. bodyweight usually caused inappetence for 48 hours. This drug was effective when given into the rumen and previous starvation did not seem to be necessary. It did not depress the egg-laying capacity of the worms.

Gordon reported that, in sheep, in doses not stated, this drug was effective against *H. contortus*, and all the species living in the large bowel, but not against *Trichostrongylus colubriformis;* it reduced the egg counts of *Ostertagi* spp. in 5 sheep by 40 to 95 per cent. when it was injected into the rumen. It was also highly effective against *Trichuris* spp., which are not affected by other anthelmintics commonly used, and against *Chabertia ovina*.

Piperazine Compounds

Riek treated 46 calves with piperazine hydrate and piperazine-1-carbodithioic betaine given by the mouth without previous starvation. Premedication with sodium carbonate did not increase its efficacy. Piperazine hydrate (3 g. per 100 lb. bodyweight) was highly effective against *O. radiatum*; at 5 g. per 100 lb. bodyweight it was effective against *O. ostertagi*; but even at 30 g. per 100 lb. bodyweight was not effective against *H. placei*, *T. axei*, *Cooperia* spp., or *B. phlebotomum*.

Piperazine-1-carbodithioic betaine, given by mouth was not more effective, at comparable dose-rates, than piperazine hydrate; but 20 g. per 100 lb. bodyweight given directly into the abomasum was highly effective against *H. placei* and *Cooperia* spp., its increased efficacy probably being due to the liberation of carbon disulphide. Gordon mentioned literature showing that, in sheep, piperazine hexahydrate and glycopiparsol were not effective against "trichostrongyles" or *Nematodirus*, *Strongyloides*, or *Trichuris*, but that, on the other hand, piperazine citrate given in the drinking water (450 mg. per kg. bodyweight for 48 hours) effectively removed *Nematodirus* spp., *B. trigonocephalum*, *Oesophagostomum* spp., and *Chabertis ovina* from lambs, but not *Trichostrongylus* spp. nor *Trichuris* spp. and it had only slight effects on *H. contortus*. In his own trials "piperazines" were highly effective against *O. columbianum* and *O. venulosum*, but had little effect on other species in sheep. This efficacy of the highly soluble piperazines destroyed the hypothesis that there is an association between insolubility in water and efficacy against the oesophagostomes.

Neguvon (0, 0-dimethylhydroxy-2, 2, 2,-trichlorophosphonate)

Riek treated more than 200 calves, aged 2½ to 12 months. The substance was given either as an aqueous solution or a 62.5 per cent. weight to volume emulsion. A dose-rate of 1 g. per 100 lb. bodyweight was highly effective against *O. radiatum*; and 5 g. per 100 lb. bodyweight was highly effective against *B. phlebotomum*. *C. pectinata*, *C. punctata*, *C. oncophora*, and *T. axei*. It was effective when it was given either by the mouth or directly into either the rumen or abomasum, except that to get good results against *Cooperia* spp. it was necessary to inject it directly into the abomasum. It was also effective against the immature stages of *H. placei*, *O. radiatum*, *B. phlebotomum*, and *Cooperia* spp., especially when 5 g. per 100 lb. bodyweight were given directly into the abomasum. At the recommended dosage the drug acted by expelling the worms, not by suppression of the egg-laying. At 5 g. per 100 lb. bodyweight there was some toxicity (increased respiration, salivation, restlessness, and looseness of the stools), but these passed off in 2 hours. Out of 200 calves treated, one calf, given 10 g. per 100 lb. bodyweight, collapsed 30 minutes after treatment and died in 60 minutes.

For the treatment of sheep Neguvon has not proved so promising. A dose of 2.5 g. per 100 lb. bodyweight was highly effective against *H. contortus* in sheep and its efficacy was, as it is in cattle, independent of the oesophageal reflex. A dose of 5 g. per 100 lb. bodyweight was effective against *T. colubriformis*, but only when it was injected, or swallowed into, the abomasum; and 10 g. per 100 lb. bodyweight given into the rumen was not effective. Efficacy against *O. columbianum* was very variable, even at dose-rates that were fatal to some sheep (10 g. per 100 lb. bodyweight).

Asuntol (Muscatox, Bayer L 21/199)

Riek gave this as a 30 per cent. wettable powder to 76 calves. The maximum safe dose for cattle is 0.25 g. per 100 lb. bodyweight which was highly effective against *H. placei*, *C. pectinata*, and *C. punctata*; and it effectively controlled *O. radiatum* in most calves. It was not effective against *T. axei* and *B. phlebotomum*. Doses up to 2 g. per 100 lb. bodyweight were tried, but these caused deaths. Gordon found this drug highly effective against *H. contortus*, *T. colubriformis*, and *T. axei* and *O. columbianum* (doses not stated) when it was injected into the rumen; but 0.25 g. per 100 lb. bodyweight was sometimes fatal and it appears to be too toxic for sheep.

Dow E.T.57 (Trolene, Viozene)

Riek gave this as a 25 per cent. wettable powder to 27 calves, the dose being 5 g. per 100 lb. bodyweight. This was highly effective against *H. placei*

and *Cooperia* spp., and sometimes against *O. radiatum*, but not against *T. axei* or *B. phlebotomum*. Gordon found it much less effective in sheep than Neguvon and Asuntol, reporting that 5 g. per 100 lb. bodyweight given into the rumen was not effective against *H. contortus*, *O. columbianum*, *T. axei*, or *T. colubriformis*, though 10 g. per 100 lb. bodyweight given into the rumen was effective against *H. contortus* and, injected into the abomasum, moderately so against *T. colubriformis*.

Phenothiazine

This was highly effective (doses not stated) against *H. placei* aged 4, 8, and 40 days, but not against worms of this species 14 days old, *i.e.* in the early 5th or late 4th larval stages. He believed also that it delayed the development of resistance (immunity) and that the only experimental cattle, in which heavy infestations were established, were those that had been treated each month with phenothiazine. Riek thinks that mature worms susceptible to phenothiazine exert some influence on resistance "thereby permitting the resistant stages to survive in large numbers."

Gordon, discussing this, thought that presumably any other highly effective anthelmintic would have a similar effect; but he had found that repeated treatment with phenothiazine had no effect on the development of resistance by lambs to *T. colubriformis*. Some of the limitations of phenothiazine as an anthelmintic for sheep have, he said, been explained by work on particle size and it appears that phenothiazine preparations should contain a proportion of very fine particles to give efficiency against *Trichostrongylus* spp. and *Ostertagia* spp. but include some coarser particles to give efficiency against *Oesophagostomum* spp. and *C. ovina*, which live in the large bowel. Thomas and Elliott (1957. *New Zealand Vet. J.* **5.** 66) suggested that the ideal preparation, from the manufacturing and anthelmintic points of view, should contain 70 per cent. of particles under 5 μ or 90 per cent. under 10 μ, the rest being up to 30 μ. Gordon thought that the influence of particle size on the toxicity of phenothiazine needs further study, as does the anthelmintic action and toxicity to sheep of phenothiazine of very fine particle size. He quoted work done in the United States which showed that some strains of sheep nematodes given phenothiazine-salt mixture for 10 years had become partially resistant to phenothiazine.

Gordon discussed the following drugs not tested by Riek:—

Cyanine Dyes

These were developed for the treatment of human filariasis. Gordon quotes some of the literature on them; other papers on them are summarised in the *Tropical Diseases Bulletin*. The cyanine dye, dithiazanine (3-3′ diethylthiodicarbocyanine) has been found by workers quoted to be highly effective against ascarids, whipworms, and trichurids of dogs and promising against *Trichuris trichiura* of man. At the McMaster Laboratory doses of 5 to 10 g. (0.2 to 0.4 g. per kg. bodyweight) showed in sheep promising efficacy against *T. colubriformis*, *H. contortus*, *and O. columbianum*.

Bephenium Compounds

Discussing the literature of these, Gordon said that they appear to be especially effective against *Nippostrongylus muris*, *Nematospiroides dubius*, *Nematodirus* spp., *H. contortus*, *T. axei*, *C. curticei*, and *Ostertagia* spp. One of them was tested at the McMaster Laboratory, *i.e.* (di-(N-benzyl-N:N-dimethyl-N-2-phenoxyethyl ammonium)-3-hydroxy-2-naphthoate). Injected into the rumen 0.5 g. of this per kg. bodyweight was highly effective against *H. contortus*, *T. axei*, and *O. columbianum*; but 2 g. per kg. bodyweight injected into the rumen was not effective against *T. colubriformis*, although injection of 0.5 g. per kg. bodyweight into the abomasum was effective in most sheep. Bephenium embonate has been found effective against *Nematodirus* in sheep.

Dithiocarbamates

Butyl-N-phnyldithio-carbamate was found by Leiper (*Vet. Rec.* **70.** 273) to be highly effective against *Nematodirus* spp. in sheep and *Ascaridia galli* in hens, but not against other trichostrongyles in sheep. At the McMaster Laboratory it was found that 6 g. of it (0.25 g. per kg. bodyweight) given into the rumen had very variable effects on *T. colubriformis*. Five g. given by mouth was effective against *H. contortus* and *O. columbianum* in 5 out of 10 sheep, a result which suggested that the drug was effective only when it was swallowed into the abomasum. This dose was effective against *Trichostrongylus* spp. in only 2 out of 10 sheep. Leiper had found that the related compound butyl-N-phenyl-thioncarbamate was effective against *Nematodirus* spp. and active against *Chabertia* and *Trichuris*. Thus the dithiocarbamates and bephenium compounds seem to be effective especially against *Nematodirus* spp. and show promise against other species in sheep.

Papain

This, Gordon considered, has a very limited value as an anthelmintic for sheep.

Carbon Tetrachloride

Work at the McMaster Laboratory has confirmed the results of other workers quoted, who found that an intramuscular injection is as effective against *Fasciola hepatica* in sheep and cattle as is adminstration by mouth. The doses given at the McMaster Laboratory were based on 1 ml. per 10 kg. bodyweight with a maximum of 30 ml. for cattle and more than 130 cattle have been thus treated without toxic effects.

Copper Methyl Arsenate (Monekil S)

This has been found at the McMaster Laboratory to be effective against *Moniezia* spp. and it reduced the number of *H. contortus*, but its toxicity to sheep is a disadvantage.

G. L.

News and Comment

OPENING OF FOUR NEW WELLCOME RESEARCH LABORATORIES

Sir Henry Dale, O.M., F.R.S., Chairman of the Wellcome Trust, was introduced by Dr. D. W. Adamson, the Research Director of the Wellcome Foundation, at the official opening of stage two of the 5-year development plan for the Wellcome Research Laboratories at Beckenham on May 1st. This major development programme was begun in 1955 to provide

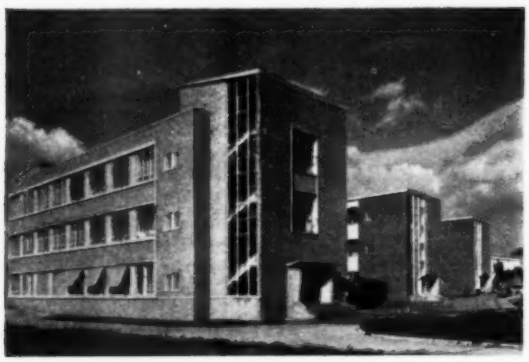

FIG. 1.—The three new virus buildings showing a part of the building of anaerobic bacteriology in the distance.

up-to-date facilities for the Wellcome Foundation's work in the field of research and the production of biological products. As Sir Henry pointed out, it was being inaugurated on that day as a going concern with 4 laboratories devoted to the production of poliomyelitis vaccine, virus vaccines and virus research work, and anaerobic vaccines. The poliomyelitis vaccine building was completed in January, 1957, and was now in full production; the party was now invited to tour the three new buildings which completed stage two of the plan.

In view of the demands for special sterility, particularly in the case of the production of virus vaccines from tissue culture, the party saw the many interesting and novel features which have been incorporated in the buildings. "Inter-storey voids" have been provided to carry the necessary engineering equipment and the working rooms are virtually free from exposed pipes. Moreover, since research and development on virological products is a continuing activity, the buildings have been constructed in such a way that they can be converted very rapidly, and separate air-conditioning plants for individual sterile areas obviate the risk of cross infection.

The first of the two new virus buildings has been provided for the production of canine distemper vaccine and canine hepatitis vaccine, as well as for yellow fever and influenza vaccine. In these laboratories the egg-adapted vaccine "Epivax" is now being prepared.

The second new virus building houses the main research and development sections for veterinary and medical work. Apart from the poliomyelitis vaccine safety testing laboratory, it consists principally of individual laboratories and offices for the study of viruses and virus diseases, such as distemper and canine virus hepatitis, and also organisms of the pleuropneumonia group. Some important work is also being carried out on the development of tissue culture which, it is hoped, will gradually take the place of the laboratory mouse, ferret, and monkey for the all-important potency tests.

The department of anaerobic bacteriology is housed in the third of the new buildings. Here the production of anaerobic vaccines is carried out side by side with relevant research work, and two separate wings provide additional space for the sterilisation of apparatus, the housing of experimental animals, and the large incubator rooms and cold store. The vaccines now in production are designed to protect against a variety of animal diseases, mainly sheep, and include lamb dysentery, pulpy kidney disease, black disease, blackleg, blackquarter, braxy, and enterotoxaemia.

The foundations of two further laboratories have already been begun, and these two buildings will complete the development scheme: one large building will be used for immunological research and for the production of a variety of biological products, whilst the second will house the new pharmacology laboratory. Mr. M. W. Perrin, Chairman of the Wellcome Foundation Limited, indicated that the Company would be spending £1,000,000 annually on research;

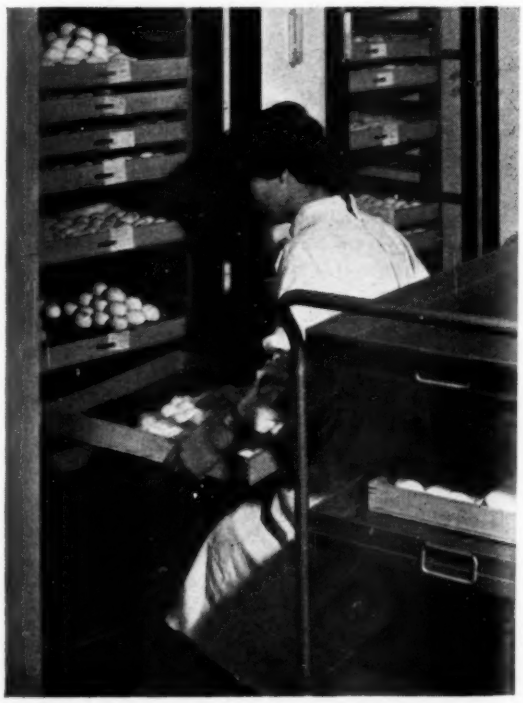

FIG. 2.- Incubation of eggs used in distemper vaccine production.

this in itself was no guarantee of success, and in thanking those who were taking part in the opening ceremony he reminded them that the Foundation's good fortune lay in the calibre of its research workers. The relationship of The Foundation with its shareholders, the five Trustees of Sir Henry Wellcome's will under the Chairmanship of Sir Henry Dale, himself once a member of the Wellcome Research team, is unusual in that all the profits passed to the trustees are distributed for the advancement of medical and veterinary science.

THE LAW RELATING TO EXPERIMENTS ON ANIMALS

Research workers will wish to know that the Research Defence Society has just published a second edition of its booklet, "Notes on the Law Relating to Experiments on Animals in Great Britain." The first edition, published in August, 1950, was intended to simplify the task of obtaining licences and certificates for animal experiments, and to obviate delays which are likely to occur when incorrect applications are made to the Home Office. It received a warm welcome from all whose work had to do with experimental animals.

The second edition has been largely re-written and, where necessary, brought up to date. Three main changes in Home Office practice have taken place during the last 8 years. They have to do, respectively, with the interpretation of Section 4 of the Act (curare); with the taking of cinematographic records of experiments; and with the licensing of technicians. The present practice has been incorporated in this second edition. Copies are free to members of the Society and the price to others is one shilling and sixpence. This would seem to be an essential publication for all research workers in the veterinary field. The Society's address is: 11, Chandos Street, London, W.1.

THE INTERNATIONAL VETERINARY CONGRESS

Members of the profession who are going to Madrid may like to be reminded that it is no longer necessary for holders of British passports to possess an entry *visa*.

ANNUAL DINNER OF THE ASSOCIATION OF STATE VETERINARY OFFICERS

The Association held its annual dinner at the Café Royal on April 25th, and about 120 members and guests attended this very pleasant function.

The toast of the B.V.A. was proposed by Mr. A. M. Taylor, President of the A.S.V.O., and Mr. H. F. Hebeler, President of the B.V.A., replied. The toast of "The Guests" was proposed by Mr. J. Edwardson, and the replies were made by Messrs. J. N. Ritchie, S. Mayne, and G. N. Gould.

Among the other guests were Dr. A. W. Stableforth, Dr. I. A. Galloway, Mr. C. P. Quick, and Mr. J. Lyons.

AN OFFER OF HELP

A Spanish veterinary surgeon who has charge of a large animal hospital attached to the veterinary school at Zaragoza, where he is also a lecturer, wishes to join a practice in Britain for the period July 22nd to August 29th. He speaks and writes English, has considerable experience of practice, and holds the Spanish doctorate of veterinary medicine. If any readers would like to have this gentleman's professional help they are asked to write direct to: Dr. Miguel Abad Gavin Pano y Ruata 9-30 A Zaragoza, Spain.

MR. RODERICK MACGREGOR

Mr. W. E. Lancaster writes:—

To those who, like myself, enjoyed the ready hospitality and the willing help and guidance of "Rory" Macgregor in their early years in the Colonial Service in Malaya, the announcement of his death on April 16th will have brought a shocked sadness. And there are many others abroad, European and Asian, who will mourn the passing of an old friend when the news eventually reaches them.

Roderick Macgregor qualified at the Royal Veterinary College in 1923 at the comparatively late age of 28 after serving in the First World War. After a brief period in general practice he joined the Malayan Veterinary Service in July, 1924; at that time the Veterinary Department was still a branch of the Medical Department. He served in several of the Malay States but mostly in Pahang where his name will always be remembered for the work he did towards restoring the buffalo herds so seriously depleted by the disastrous floods in that State in 1926. He had a great appreciation of the place of the domestic buffalo in Malaya's rural economy and made a special study of this animal, gaining his Fellowship for his thesis on it in 1939. He became, to use his own words, a "fanatical buffalo lover" and seldom missed an opportunity of extolling its virtues.

When Malaya fell to the Japanese in 1942 Macgregor was on leave out of the country and he continued his Colonial service during the war years in Palestine and Nigeria. At the end of the war he returned to Malaya with the rank of major in the British Military Administration but retired from the Service in 1946 on reaching the age limit. At the National College of Food Technology, London, where he subsequently obtained employment, he taught meat inspection, a subject on which he wrote a students' handbook.

"Rory" had a quiet, mild manner that concealed a firm character and strength of purpose. He enjoyed company and conversation and his dry humour and gift for an apt Biblical phrase were always a source of delight to those who knew him.

PERSONAL

Dr. Charles G. Durbin has been appointed veterinary medical director of the U.S.A. Food and Drug Administration's bureau of medicine. He succeeds the late Dr. John H. Collins.

Births

BOX.—On April 28th, 1959, at The Chestnuts, Bury St. Edmunds, to Paula, wife of Philip G. Box, M.R.C.V.S., a son, Duncan Peter, brother for Nigel.

PALMER.—On May 2nd, 1959, at the Maternity Hospital, Mill Road, Cambridge, to Shirley, wife of Dr. A. C. Palmer, M.A., M.R.C.V.S., a son.

UNIVERSITY NEWS

Bristol

Third Examination for the Degree of B.V.Sc.
April, 1959

PASS

Anstey, Jean Mary; Blake, Kenneth John; Burrows, Charles John; Davies, Hugh Emlyn; Gregory, Martin Wicksteed; Jenkins, Charles Trevor; Morris, John Malcolm; Peace, Michael David; Perry, Michael David; Sebesteny, Andor; Skelton, William Ewbank; Turner, Peter John; Westcott, Mary; Whittard, Lawrence Mervyn; Wilkinson, Keith Ronald; Young, Robert Charles.

COMING EVENTS

May

12th (Tues.). Meeting of the East Midlands Division at the University School of Agriculture, Sutton Bonington, 2.15 p.m.

13th (Wed.). General Meeting of the Lancashire Veterinary Association at Booth's Café, 5, Fishergate, Preston, 2.30 p.m.

Spring Meeting of the Eastern Counties Veterinary Society at the Angel Hotel, Bury St. Edmunds.

Annual General Meeting of the West of Scotland Division in the Grand Hotel, Sauchiehall Street, Glasgow, 2.30 p.m.

15th (Fri.). Dance of the V.V.B.F. Ladies' Guild Eastern Counties Division at the Guildhall, Cambridge, 8 p.m.

Meeting of the B.V.A. Welsh Branch in the Geography Department, University College of Wales, Alexander Road, Aberystwyth, 2.15 p.m.

The 167th Annual Ball of the Royal Veterinary College at the Park Lane Hotel.

21st (Thurs.). Annual General Meeting of the Society of Practising Veterinary Surgeons in the Midland Hotel, Manchester, 2.30 p.m.

Ordinary General Meeting of the Royal Counties Veterinary Association in the Education Lecture Room, Reading University, 3.15 p.m.

27th (Wed.). Annual General Meeting of the Sussex Veterinary Society at the Old Ship Hotel, Brighton, 2.30 p.m.

28th (Thurs.). Annual General Meeting of the Supplementary Veterinary Register Division at "The Crown," Brewer Street, Piccadilly, 3.30 p.m.

June

2nd (Tues.). R.C.V.S. Day. Annual General Meeting at the Royal College of Veterinary Surgeons, 10, Red Lion Square, London, W.C.1, 3.45 p.m.

3rd (Wed.). Annual General Meeting of the Victoria Veterinary Benevolent Fund at 10, Red Lion Square, London, W.C.1, 1.30 p.m.

3rd and 4th (Wed. and Thurs.). Quarterly Meetings of Council of the R.C.V.S.

4th (Thurs.). Meeting of the Western Counties Veterinary Association at the Rougemont Hotel, Exeter, 2.30 p.m.

11th (Thurs.). Annual Meeting of the R.A.V.C. Golfing Society at the North Hants Golf Club, Fleet, 10 a.m.

12th (Fri.). Annual Dinner of the R.A.V.C., Officers' Club at Grosvenor House.

13th and 14th (Sat. and Sun.). 7th Annual Reunion and Dinner of the R.V.C. '52 Club at Streatley.

July

15th, 16th and 17th (Wed., Thurs. and Fri.). B.V.A. Committee and Council Meetings in London.

August

30th to 5th September. 77th Annual Congress of the British Veterinary Association in Folkestone. (Official opening Monday, August 31st.)

ADDRESSES OF DISEASE INFECTED PREMISES

The list given below indicates, first the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak.

Anthrax

Arran. Mount Pleasant Farm, Lamlash, Brodick (Apr. 27).
Ayr. Dalvennan Farm, Maybole (May 4).
Isle of Wight. Chine House, Brook Green, Newport (Apr. 28).
Lanarks. Auchnotroch Farm, Auchenheath (May 4).
Lancs. Woodstraw Farm, Thornley, Longridge, Preston (Apr. 27).
Northants. Manor Farm, Woodford, Kettering (May 1).
Salop. Shavington Home Farm, Market Drayton (Apr. 30).
Stirling. Cushenquarter Farm, Plean (Apr. 27).

Fowl Pest

Dorset. Court Orchard, Stoke Abbott, Beaminster; Potwell Farm, Mosterton, Beaminster (Apr. 27); Primrose Cottage, Solway Ash and Slaughterhouse, Solway Ash, Bridport; Brook House, Gondover Loders, Bridport; New House Farm, Loders, Bridport (Apr. 30); Crabb Orchard Farm, Three Legged Cross, Wimborne (May 1); Brook House, Upper Loders, Bridport (May 4).
Essex. Walscot Farm, Broomfield, Chelmsford (Apr. 30).
Lancs. New Hall Farm, Tyldesley, Manchester (Apr. 27); Premises, Orrell Lane, Burscough, Ormskirk (Apr. 28).

Swine Fever

Cambs. Glebe House Farm, Landwade, Fordham, Ely (Apr. 27); Ravens Hall Farm, Burrough Green (Apr. 28).
Ches. Crewe Hall Piggeries, Farndon (Apr. 27); Lower Hall Farm, Picton (Apr. 28); Lower Erncroft, Marple Bridge, Stockport; Heath Farm, Burton, Wirral (Apr. 30).
Devon. Maldon Farm, Okehampton (Apr. 27); Dowrich Farm, Sandford, Crediton (Apr. 29).
Denbigh. Gwen-y-Fran, Maerdy, Corwen (Apr. 29).
Derby. Grange Farm, Stanley (Apr. 30); 68, The Bottoms Conningdale, Fairfield, Buxton (May 4).
Essex. Maythorne, Hovefields Avenue South, North Benfleet, Basildon (Apr. 27); Field Gate, West Hanningfield, Chelmsford (Apr. 29); Suttons Farm, Bird Lane, Upminster (May 4).
Glamorgan. The Droces, Penarth Road, Cardiff (Apr. 27).
Lancs. Five Rhodes Green, Rhodes, Middleton (May 4).
Leics. Groby Park Farm, Markfield (May 4).
Norfolk. Lodge Farm, Horsford, Norwich; Galleymoor Farm, East Dereham (Apr. 30); Station Road, North Elmham, Dereham (May 4).

Northants. Buildings Farm, Newborough, Peterborough (May 4).

Notts. Dennis Street Gardens, Netherfield (Apr. 29).

Somerset. Broad Street Farm, Stratton, Evercreech, Shepton Mallet (May 4).

Surrey. Woodley, Selsdon Vale, Sanderstead (May 4).

Warwicks. College Farm, Willington, Shipston on Stour; Pincock Farm, Oxhill (Apr. 27); Cornetts End Farm, Berkswell, Coventry (Apr. 28); Model Farm, Harbary, Leamington Spa; Upperskilts Farm, Mappleborough Green (Apr. 30); Thatched Cottage, Avon Dassett, Leamington Spa (May 4).

Yorks. Crompton Farm, Haxby (Apr. 29).

AGRICULTURAL STATISTICS (ENGLAND AND WALES): ESTIMATED NUMBERS OF LIVESTOCK

Description	Thousands March 1958	March 1959
Cows and heifers in milk :—		
For producing milk or calves for the dairy herd	2,134	2,032
Mainly for rearing calves for beef	303	289
Cows in calf but not in milk :—		
Intended for producing milk or calves for the dairy herd	473	498
Intended mainly for rearing calves for beef	214	215
Heifers in calf (first calf)	600	633
Bulls being used for service	60	56
Bulls (including bull calves) being reared for service	22	23
Other cattle :—		
Two years old and over :—		
Male	503	506
Female	432	386
Total	(935)	(892)
One year old and under two :—		
Male	562	695
Female	973	974
Total	(1,535)	(1,669)
Under one year old :—		
Male	716	833
Female	1,147	1,165
Total	(1,863)	(1,998)
TOTAL CATTLE AND CALVES	8,140	8,306
Ewes in lamb or with lambs at foot	7,491	7,932
Other sheep over 6 months old (excluding rams for service)	2,812	3,396
Sows in pig	336	312
Gilts in pig	139	84
Other sows kept for breeding	207	177
Total sows for breeding	(682)	(572)
Barren sows for fattening	22	23
Boars being used for service	40	35
All other pigs (not entered above) :—		
Five months old and over	947	1,027
Two months old but not yet five months old	1,992	2,005
Under two months old	1,410	1,263
TOTAL PIGS	5,093	4,925
Fowls :—		
Six months old and over	36,893	37,995
Under six months old :—		
Male	4,874	4,788
Female	21,437	23,697
Sex not known	2,433	4,531
Total	(28,744)	(33,017)
Total fowls	(65,637)	(71,012)
Ducks of all ages	721	681
Geese of all ages	142	125
Turkeys of all ages	321	427
TOTAL POULTRY	66,821	72,244

General Summary

The results of the March 1959 Agricultural Returns for England and Wales show some reduction, as compared with March 1958, in the number of cows on farms but there are more calves than a year ago. There have been increases over the year in sheep for breeding and in fowls, and a decrease in the pig breeding herd.

Cattle

The total breeding herd (cows of both beef and dairy types) is 90,000 (3 per cent.) below the March 1958 figure, but there has been an increase of 33,000 (5½ per cent.) in the number of heifers in calf (first calf).

There are 136,000 (7 per cent.) more calves on farms than at March 1958.

Sheep

The sheep breeding flock is up by 441,000 (6 per cent.) over March 1958.

Pigs

The March 1959 total for sows and gilts for breeding is 110,000 (16 per cent.) below the peak level of March 1958, and 6 per cent. below the December 1958 level.

Poultry

There are about one million (3 per cent.) more fowls of 6 months old and over than a year ago.

The total number of fowls under 6 months old is 4¼ million (15 per cent.) above the level of March 1958, increases coming from female birds (up by 10½ per cent.) and from the total of male birds and unsexed chicks (up in total by 28 per cent.), which suggests further increases in table poultry production as well as more entrants to next year's laying flock.

AGRICULTURAL STATISTICS ADVISORY COMMITTEE

The Minister of Agriculture, Fisheries and Food has appointed Mr. J. H. Kirk, C.B.E., one of the Ministry's Under Secretaries, to be Chairman of the Committee in succession to Mr. J. A. Sutherland-Harris. The main duties of the Committee are to advise the Minister on the collection of statistical returns from farmers and related matters.

The Minister has also appointed Mr. C. J. Brown, Chief Statistician and head of the Statistics Division of the Ministry, to be an additional member of the Committee.

ADVERTISER'S ANNOUNCEMENT

BURROUGHS WELLCOME & CO. announce the issue of "Franocide" brand Diethylcarbamazine Injection (Veterinary) for the control of parasitic bronchitis. "Franocide" is available in bottles of 400 c.c., sufficient for the treatment of four average-sized calves. Its issue will be confined to members of the veterinary profession or on their prescription.

Letters to the Editor

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the B.V.A.

Treatment of Skin Infections in Dogs

Sir,—I have been using a new product, "Hexetidine,"* which has been marketed for the treatment of fungal skin infections and for various forms of non-specific dermatitis.

At the time this product was delivered to me I was attempting to treat and control an outbreak of ringworm in a large kennel of greyhounds. The treatment I had previously adopted was by means of various brands of proprietary ointments, which, whilst curing individual lesions, did not prevent the appearance of new lesions on the affected animal nor its spread to other dogs.

Hexocil is applied to the entire skin surface in the form of a shampoo and the coat is then thoroughly rinsed after 2 hours.

Eight animals were treated and in each case there was complete recovery after one application, and after a period of some 4 weeks no new lesions have occurred.

A number of animals with single lesions were treated with local applications only and these have similarly responded to treatment. So far no new cases have been reported.

The advantage of this product from the point of view of the general practitioner is the ease of application and the fact that the whole body area can be treated at one time.

Yours faithfully,
ERIC W. FAWCUS.

The Surgery,
230, South Street,
Romford,
Essex.
May 1st, 1959.

* Hexocil, William Warner & Co., Ltd.

The R.C.V.S. Council Elections

Sir,—A number of requests from different sources ask candidates for election to the Council of the R.C.V.S. to state their views before electors cast their votes. Such requests are reasonable in any democratic organisation.

The Veterinary Surgeons Act, 1948, brought about changes in the duties and constitution of the R.C.V.S., so that outside bodies such as the Privy Council and the Universities can nominate representatives to sit on the Council. The duties of the R.C.V.S. now resemble those of the General Medical Council; in the main, the R.C.V.S. is responsible for educational standards and professional ethics in accordance with the requirements of the times. These twin tasks are not small or simple and will demand great courage in future negotiations with governmental and public bodies. I consider that one of the most urgent tasks of the R.C.V.S. in education is to develop post-graduate facilities, especially for clinical subjects within the veterinary schools.

There need be no confusion as to the relative duties of the R.C.V.S. and the British Veterinary Association. The consideration of professional salaries comes within the purview of the B.V.A. but consideration of professional fees might require amicable liaison between R.C.V.S. and B.V.A. for the question of ethics could be involved.

With regard to the question of disease control, I believe that for many reasons this must remain the responsibility of the Government. It is essential, however, that in formulating policy the veterinary profession be fully consulted as the experts in matters concerned with animal health and its effect upon public health.

I believe that a great future lies before us and our representative bodies will need the thoughtful support of each one of us in order to fulfil their wide functions.

Yours faithfully,
FRED R. BELL.

Royal Veterinary College,
Royal College Street,
London, N.W.1.
May 5th, 1959.

DISEASES OF ANIMALS ACT, 19 o—GREAT BRITAIN

Summary of Returns of Confirmed Outbreaks of Scheduled (Notifiable) Diseases

Period		Anthrax	Atrophic Rhinitis	Foot-and-Mouth	Fowl Pest	Sheep Scab	Swine Fever
1st to 15th April, 1959		20	—	—	42	—	49
Corresponding period in	1958	3	1	8	28	—	43
	1957	31	—	—	18	—	29
	1956	70	—	—	22	—	30
1st Jan. to 15th April, 1959.		83	—	1	260	—	337
Corresponding period in	1958	55	3	108	223	—	311
	1957	164	2	15	269	—	242
	1956	892	3	8	193	—	183